

Torsade de pointes

Sir,

In an Editorial (*British Heart Journal*, 1976, 38, 117) the authors recommend intravenous isoprenaline infusion as the first step for the treatment of *torsade de pointes* ventricular tachycardia. This has the theoretical basis of shortening the repolarisation time and thus avoiding a state of asynchronous depolarisation. Drugs which increase the repolarisation time, such as quinidine, are not only not indicated for the treatment of the above arrhythmia, but can induce it. Though, theoretically, isoprenaline is an ideal agent, there is a risk of inducing ventricular arrhythmias, especially in patients with myocardial ischaemia. This may also occur with the slight increase of the optimal serum concentration of the drug. On the other hand, isoprenaline as a beta-adrenergic stimulant can induce the side effects of sympathomimetics.

Torsade de pointes ventricular tachycardia has been observed (Kounis, 1976) in a patient on prenylamine who was suffering from syncopal attacks and who was given slow release isoprenaline hydrochloride tablets (Saventrine 30 mg t.i.d.) by his general practitioner because he was thought to have Adams-Stokes attacks. Though prenylamine had been discontinued, the patient continued to have syncopal attacks while he was taking Saventrine for the next 7 days. After his admission he was found to have *torsade de pointes* ventricular tachycardia with prolongation of the QT interval. This was attributed to Saventrine because sometimes sympathetic stimulation prolongs the QT interval (Yanowitz *et al.*, 1966). Mexiletine, however, might be a good and effective first step and if isoprenaline is preferred, cardiac pacing must always be instituted simultaneously.

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References

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Yanowitz, F., Preston, J. B., and Abildskov, J. A. (1966). Functional distribution of right and left stellate innervation to the ventricles: production of neurogenic electrocardiographic changes by unilateral alteration of sympathetic tone. *Circulation Research*, 18, 416-428.

This letter was shown to Drs. Krikler and Curry who reply as follows:

Sir,

Dr. Kounis quite properly raises queries about the management of this disorder. In our Editorial (Krikler and Curry, 1976) we mentioned its rarity in cardiac ischaemia, citing the explanation offered by Puech (1974); when it occurs in this setting it may well be the result of complicating paroxysmal atrio-ventricular block (Chiche *et al.*, 1974), and we wonder whether this, or sinoatrial block aggravated by prenylamine (Evans, 1975) was a factor, in addition to the direct effects of prenylamine on repolarisation.

Failure to respond to oral isoprenaline does not mean that this agent directly aggravated the situation: indeed, its effect in accelerating the heart rate and shortening the QT interval may have been inadequate. While the QT interval may be prolonged by the *rapid* intravenous injection of isoprenaline, it is shortened by *infusion* (Abildskov, 1976), a paradox explored in this recent paper. Thus our therapeutic advice is not contradicted by the continuation of the work quoted by Dr. Kounis (Yanowitz *et al.*, 1966). We agree with the inference that pacing may be preferable, but this may not necessarily be available where the patient presents, and, if underlying factors (other than heart block) remain uncorrected, *ventricular* pacing may actually induce *torsade de pointes* (Evans *et al.*, 1976).

Mexiletine has indeed proved useful in the prevention of recurrent *torsade de pointes* after the precipitating factors had been treated (Curry *et al.*, 1976); perhaps it suppresses extrasystoles that could initiate episodes. We have, however, seen three further cases where it occurred when lignocaine was infused after myocardial infarction in patients who had become hypokalaemic because of vigorous

diuretic therapy. Under other circumstances the infusion of lignocaine or mexiletine may well help the occasional case, but the value of treatment with intravenous isoprenaline and of atrial pacing has been shown (Slama *et al.*, 1973).

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Notice

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